Case Report

Thyrotoxicosis-induced cardiomyopathy treated with venoarterial extracorporeal membrane oxygenation

Ivo Geneva, Michelle D. Lundholma, Mary Ann Emanuele, Edwin McGee, Verghese Mathewed,*

*Department of Internal Medicine, Loyola University Medical Center, Maywood, IL, USA
bDepartment of Medicine, Division of Endocrinology, Loyola University Medical Center, Maywood, IL, USA
cDepartment of Medicine, Division of Thoracic and Cardiovascular Surgery, Loyola University Medical Center, Maywood, IL, USA
dDepartment of Medicine, Division of Cardiology, Loyola University Medical Center, 2160 S First Ave, Suite 6232-6234, Maywood, 60153 IL, USA

Keywords:
Hyperthyroidism
Extracorporeal membrane oxygenation
Graves' disease
Cardiomyopathy

Article history:
Received 16 May 2019
Received in revised form 16 June 2019
Accepted 24 June 2019
Available online xxx

A B S T R A C T

A 37-year-old woman with no past medical history presented with longstanding untreated hyperthyroidism and consequentially developed thyrotoxicosis-induced cardiomyopathy. Upon admission, she was noted with a heart rate of 172 beats per minute and an EKG consistent with supraventricular tachycardia which was unresponsive to adenosine. Shortly after the initiation of a non-cardioselective beta-blocker for the treatment of persistent tachycardia, she developed profound cardiogenic shock refractory to vasopressors and inotropes. She was diagnosed with thyroid storm, which was ultimately attributed to Graves' Disease and controlled with propylthiouracil, potassium iodide drops, and hydrocortisone. Extracorporeal membrane oxygenation (ECMO) was successfully used as a temporizing measure while her thyroid hormone level normalized and cardiac function recovered. Patients with longstanding untreated hyperthyroidism may be dependent on the induced hypervascular state to compensate for low-output cardiac failure, therefore it is important to exercise caution when initiating beta-adrenergic blockade. Given the expected disease time-course in cases of acute decompensation, ECMO remains a viable option for short-term mechanical circulatory support.

© 2019 Elsevier Inc. All rights reserved.

Introduction

Hyperthyroidism is a common metabolic disorder that may lead to life-threatening thyrotoxicosis and thyroid storm.1 Patients with the condition commonly present with sinus or supraventricular tachycardias, fever, central nervous system manifestations (seizures, coma, delirium, psychosis, or simply agitation), or gastrointestinal symptoms such as vomiting and diarrhea.2 Signs of heart failure, however, may also be encountered. In these cases, addressing the underlying etiology of thyroid disease becomes important in order to ultimately address the cardiac dysfunction, which if left untreated may continue to deteriorate, ultimately requiring the utilization of mechanical circulatory assist devices.3

Case report

A 37-year-old woman with no past medical history presented with 3 days of lower extremity swelling and several months of palpitations. Her heart rate was 172 beats/minute with narrow and regular QRS complexes on EKG, consistent with supraventricular tachycardia (SVT). The rest of her admission vital signs included a blood pressure of 163/116, temperature of 98.4 F, and a respiratory rate of 16 with an oxygen saturation of 98% on room air. She received two doses of adenosine, 6 mg followed by 12 mg two minutes later, without change in heart rate. On examination, she was alert and oriented. Her neck was supple without thyromegaly or nodules. No oculopathy was appreciated. Her lungs were clear to auscultation and she did not have any cardiac murmurs. She had pitting pedal edema with profound weakness of her proximal muscles. bedside echocardiography revealed a dilated and diffusely hypokinetic left ventricle and a grossly reduced ejection fraction (EF) visually estimated at 30%. Labs showed TSH < 0.01 uIU/mL (0.40–4.60 uIU/mL), total T3 of 484 pg/dL (60–116 pg/dL) with a free T3 of 887 pg/dL (171–371 pg/dL), and total T4 of 29.7 ug/dL (5.0–11.0 ug/dL) with a free T4 of 4.7 ng/dL (0.7–1.5 ng/dL). Her Burch-Wartofsky Point Scale for Thyrotoxicosis was 40, concerning for impending thyroid storm. She was admitted to the intensive care unit where she was given a diltiazem bolus and started on a drip. The following morning she was transitioned to 60 mg of propranolol three times daily due to persistent tachycardia. Two hours after the first dose, she developed severe shortness of breath and agitation. Within minutes, she was unresponsive and profoundly hypotensive to 50/25 mmHg. Her lactate increased to 6.4 mmol/L (0.4–1.3 mmol/L).
Discussion

The initial effects of triiodothyronine (T3) on nucleic thyroid hormone receptors of cardiac myocytes include upregulation of structural and regulatory genes that result in enhanced contractile force. Transesophageal echocardiogram demonstrated severely reduced EF of 10%, bowing of the interventricular septum and a decreased right ventricular function. Her tricuspid valve was wide open with a dilated annulus. She was started on venaoarterial extracorporeal membrane oxygenation (ECMO). Based on the Japan Thyroid Association and Japan Endocrine Society guidelines, she now fit the criteria for “definite” thyroid storm—elevated free T3/T4 plus agitation and a Glasgow coma scale < 14 plus tachycardia and cardiogenic shock—therefore once stabilized, she was loaded with 500 mg of propylthiouracil (PTU), potassium iodide 100 mg every 8 h. 

Ultimately, her thyroid stimulating antibody, which had been drawn on hospital day two, approximately 24 h after her initial deterioration in clinical status, returned at 9.08 IU/L (0.10 IU/L) and she was diagnosed with thyroid storm from Graves’ Disease. One week after the initiation of therapy, her free T3 had decreased to 160 ng/dl and free T4 to 1.4 ug/dl, and she no longer required inotropic or vasopressor support. Transthoracic echocardiogram noted improvement in her LVEF to 35%. She was successfully decannulated from ECMO after 8 days and subsequently extubated. She was discharged to an acute rehabilitation facility on PTU and propranolol. At her 6-month follow-up, she was able to participate in intense exercise without residual deficits and was overall doing well.

PTU and potassium iodide rapidly control thyroid hormone levels in thyroid storm and, in most cases, improve ventricular function within two weeks of initiation. Given this expected time-course, the addition of venaoarterial ECMO, in which the patient’s blood is pulled from the right atrium through an external oxygenator and returned to the femoral artery, remains a viable option for provisional circulatory support.

Expected survival to discharge on ECMO can be calculated using the Survival after Veno-Arterial ECMO (SAVE) Score. Of the 15 published cases in the literature, 12 survived to discharge with almost complete recovery of ventricular function. Predicted survival for this patient was 30% (Class IV), despite which, she did well with eventual return to baseline functional status. This case adds to a handful of others in the literature where ECMO was successfully utilized for up to 18 days in the setting of severe thyrotoxicosis-induced cardiogenic shock despite maximal inotropic support.

Conclusion

In summary, caution must be exercised when escalating beta-adrenergic blockade in patients with thyroid storm and suspected thyrotoxicosis-mediated cardiomyopathy. Identification of the underlying etiology of the thyroid dysfunction is important in long-term management. In acutely decompensating patients with refractory cardiogenic shock, ECMO should be considered for temporary circulatory support. This is especially true in younger patient populations where survival to discharge appears to be higher than initially predicted.

References

14. ELSO Adult Cardiac Failure Supplement to the ELSO General Guidelines Extracorporeal Life Support Organization (ELSO) Guidelines for Adult Cardiac Failure; 2013.